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Short-Term Nitrogen Dioxide Exposure and Acute Respiratory Disease in Children

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A CHESS data base from Chattanooga, Tennessee was thoroughly scrutinized and found to be of high enough quality to warrant epidemiological analysis. Using this data base, the relationship between NO_2 ambient pollution levels and acute respiratory disease in children was examined. Although a statistically significant relationship was found, it was not monotonic. Indeed, over the range of pollution values experiences, more illness is associated with low pollution values than with high ones. A U-shaped relationship between illness and NO_2 concentrations was found in several subpopulations in addition to the entire data set, although for some subpopulations no relationship was found. In contrast, higher ambient sulfate levels were found to have a positive effect on acute respiratory disease incidence in children over the entire period and for different subsamples, although this effect was not significant for either season analyzed separately.

Since the passage of the Clean Air Act in 1970, several epidemiological studies have attempted to associate morbidity with indoor and outdoor exposure to nitrogen dioxide (NO_2). The indoor, so-called gas stove studies[1-7] produced mixed and inconclusive results in their attempts to link health impairment to the presence of a gas stove or gas heater in the home. Studies of the health effects of outdoor NO_2 exposures also have failed to find consistently significant health effects at ambient exposure levels.[8-11]

In an analysis of people living in Chattanooga, Tennessee, conducted under the Community Health and Environmental Surveillance System (CHESS) program, Shy and Love[12] were able to link NO_2 exposures and acute respiratory disease. However, several problems have been raised about this study. The researchers have been criticized for using rudimentary statistical techniques, consisting mainly of pairwise comparison of illness incidence rates in subpopulations. In addition, the data base has been tainted by its association with the controversial CHESS program.[13] [The earlier CHESS Chattanooga studies were also criticized for using a subsequently discredited method (Jacobs-Hochheiser) for monitoring NO_2 concentrations. However, by 1972 the Saltzman technique was being used.] Yet EPA has found the Chattanooga data to be accurately transferred from the surveys to the computer tapes and our own research has revealed the data quality to be at least as high as other similar, but much less controversial data bases.

The lack of persuasive epidemiological studies upon which to base a national ambient air quality standard for nitrogen dioxide motivated the present paper. Here we return to the CHESS aerometric and health data bases collected during 1972-73 in Chattanooga and used by Shy and Love to examine the relationship between NO_2 and acute respiratory disease in children. We first describe and defend the CHESS-Chattanooga data base and the statistical model used to examine it. We then present our results and discuss a number of econometric issues and their relationship to our findings.

The Data Base

In January 1972, a self-administered survey on chronic respiratory disease (CRD) was distributed to families with children in elementary schools in one of the three Chattanooga communities, Harrison, Brainerd, and Redbank, located within one mile of an air pollution monitoring station. A subsample of families—1970 parents and their children, 4898 individuals in all—was drawn from this sample to participate in an acute respiratory disease (ARD) panel survey. Information was taken in two-week intervals (always beginning on a Sunday) over three school semesters from spring 1972 through spring 1973. Each family was phoned within several days after the end of each two-week period to determine if any family members experienced various acute respiratory disease symptoms or consulted a physician.

Aerometric data were gathered at seven sites. Hourly measurements of NO_2 were taken using the Saltzman chemiluminescence technique only for the fall 1972 and spring 1973 study periods. Thus, we eliminated data for the spring 1972 period from our analysis. Chattanooga was chosen as a site for an NO_2 study because it featured a TNT plant emitting large quantities of nitrogen-based pollutants. This plant closed January 1, 1973, resulting in reduced NO_2 concentrations in the nearby communities. Daily readings were taken on particulates, nitrates, and sulfates. These daily readings were reduced to monthly frequency distributions. Unfortunately, the original daily data were unavailable from EPA, and we have been forced to use the monthly frequency distributions for the latter three pollutants.

The Chattanooga health and aerometric data collection effort of the early 1970s and the CHESS program in general have been criticized (Roth[14]) for their poor survey protocols, health data inconsistencies, and aerometric data unreliability. Krupnick and Harrington[15] provide a complete reanalysis of these data and find, first, that the survey protocols were carefully designed and observed. In addition, responses to identical sociodemographic questions on the CRD and ARD surveys were found to be quite consistent. Also, the NO_2 monitoring data were found to be reasonably complete, generally consistent, and taken by devices that generally outperform other types of monitors in the lab.

Further, because a duplicate CRD survey was administered to some of the participants 22 months later, we were

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able to identify inconsistencies intertemporally. Drawing on responses from 948 parents and considering only the questions concerning age, race, birthdate, education, and smoking status, over 80% of the parents had matching responses over the two surveys. These results compare favorably to similar investigations of the U.S. Census and other highly regarded data bases.[16,17]

In the course of our examination of the quality of this data base we noted a number of recall-related problems inherent in the survey procedure. These problems may also be present in parts of other surveys, such as the Health Interview Survey (HIS), that rely on biweekly interviews to collect acute respiratory disease data. These problems are discussed in some detail elsewhere,[15] but the main points may be summarized as follows:

1) Respondents have imperfect recall of the day or even week of onset of illness. For example, over 60% of illnesses reported were said to have occurred in the second week of the recall period, a result significantly ($\alpha = 0.01$) different from the uniform distribution one would expect. Apparently respondents either forget (presumably minor) illnesses occurring in the first week, or they remember disease onset as occurring later than was actually the case.

2) When average duration of reported illness is plotted as a function of day of onset during the two-week period, a linear decline is found during the second week of the period, with average duration at the end of the week barely half of average duration at the beginning. The most likely explanation is that illnesses extending past the end of the period are not reported accurately, even though interviewers were instructed to identify such illnesses and ask about them at the end of the next reporting period. If this explanation is correct, the truncation of restricted activity days imparts a downward bias to illness severity.

3) In other panel studies it has been suggested that respondents may, over time, progressively under-report illness simply because they become tired of doing interviews. If pollution levels are time-dependent, the study results may be biased accordingly. We found little evidence of this phenomenon. On the assumption that less serious illnesses could be more likely to be neglected, we regressed the ratio of "serious" to total illness incidence on time, and found no trend.

These findings affected our subsequent data analysis in two ways. First, no attempt was made to use time intervals shorter than two weeks, even though the sample could be reduced to weekly or even daily observations. Second, we concentrated on the incidence of illness rather than duration, inasmuch as we felt the former to be more reliable.

Table I. Descriptive statistics ($N = 2093$).

Variable	Mean value or population fraction
NEWILL	0.13
RADS	0.21
AGE	7.7
Age distribution	
0–2	0.09
3–4	0.08
5–6	0.16
7–8	0.23
9–10	0.25
11–12	0.19
RACE1W	0.91
CHESTINF	0.28
CHRON	0.07
Education of household head	
High school graduate	0.71
Attended some college	0.45
MOMHEAD	0.05
Mothers' smoking status	
Current	0.32
Ex-	0.15
Non-	0.53
CROWD	1.30
SEX1F	0.48
GAS	0.05
RAIN	2.70
TEMP	18.20

Table II. Pollution statistics.

	Mean ($\mu g/m^3$)	Standard deviation	Correlation coefficients PAR90P	SUL90P	TEMP
NO2MAX	98.0	48.3	−0.10	0.20	−0.09
PAR90P	100.6	31.4		−0.027	0.34
SUL90P	10.0	2.7			−0.036

The Model

To identify the factors affecting reported children's disease, we use pooled cross-section time series models predicting illness incidence or duration as a function of demographic, pollution, and weather variables. Symbolically, the models are of the form

$$S_{ijt} = f(X_{ij}, P_{jt}, W_t) + \epsilon_{ijt}$$

where S_{ijt} is the reported incidence or duration of the illness of the ith child in the jth neighborhood during period t,
X_{ij} is a vector of personal variables for the ith child in the jth neighborhood,
P_{jt} is a vector of pollution variables for the jth neighborhood in period t,
W_t is the weather in period t, and
ϵ_{ijt} is the disturbance term.

The independent variables are defined as follows:

AGE: the child's age at the beginning of the school year.
RACE1W: the race of the head of household; 1 if white, 0 if nonwhite.
CHESTINF: 1 if the child has suffered a respiratory infection within the past three years, 0 otherwise.
CHRON: 1 if the child suffers from asthma or a chronic heart or lung condition, 0 otherwise.
EDU: the years of schooling completed by the head of household.
MOMHEAD: 1 if the household head is female, 0 otherwise.
SMKPPD: mother's smoking in packs per day.
CROWD: number of household members divided by the number of rooms in the house.
SEX1F: sex of child; 1 if female, 0 if male.
GAS: 1 if the kitchen stove is gas, 0 if electric.
RAIN: amount of rainfall during the period, in inches.
EPIDEM: monthly influenza cases reported by the State of Tennessee (in thousands).
TEMP: the absolute difference between the average temperature during the period and 65°.
NO2MAX: average daily maximum concentration of NO_2, in $\mu g/m^3$.
PAR90P: 90th percentile total suspended particulate concentration during the month, in $\mu g/m^3$.
SUL90P: 90th percentile sulfate concentration during the month, in $\mu g/m^3$.

As noted above, two dependent variables are considered: illness incidence (NEWILL), which is 0 or 1 according to

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Table III. Predicting illness incidence in population subsamples.[a]

	A All children 12 and under	B Children with nonsmoking mothers	C Children with smoking mothers	D Children without chronic respiratory disease	E Children with CRD	F Children 6 and under	G Infants	H Fall only	I Spring only
Intercept	0.0512	0.064	−0.0197	0.052	0.098	0.037	0.151	0.161	0.018
	(0.0503)	(0.059)	(0.091)	(0.056)	(0.095)	(0.10)	(0.21)	(0.113)	(0.075)
NO2MAXL	−8.87E-4	−6.10E-4	−13.5E-4	−6.4E-4	−13.6E-4	−8.44E-4	−12.5E-4	0.26E-4	−15.0E-4
	(2.05E-4)[c]	(2.46E-4)[b]	(3.71E-4)[c]	(2.3E-4)[c]	(4.0E-4)[c]	(4.1E-4)[b]	(8.7E-4)	(4.3E-4)	(2.9E-4)[c]
NO2MAXH	1.71E-4	2.19E-4	−.57E-4	2.3E-4	.51E-4	2.41E-4	5.2E-4	0.06E-4	3.95E-4
	(0.68E-4)[b]	(0.78E-4)[c]	(1.46E-4)	(1.13E-4)[b]	(1.34E-4)	(1.89E-4)	(2.55E-4)[b]	(0.78E-4)	(2.01E-4)[b]
PAR90P	8.88E-4	6.69E-4	14.3E-4	11.4E-4	4.00E-4	12.1E-4	13.0E-4	28.1E-4	13.4E-4
	(6.08E-4)	(7.4E-4)	(10.8E-4)	(6.8E-4)	(12E-4)	(11.9E-4)	(24E-4)	(15E-4)	(9.1E-4)
PAR90P2	−0.053E-4	−0.044E-4	−0.075E-4	−0.058E-4	−0.041E-4	−0.066E-4	0.045E-4	−0.146E-4	−0.056E-4
	(0.026E-4)[c]	(0.03E-4)	(0.048E-4)	(0.031E-4)	(0.0555)	(0.054)	(0.11E-4)	(0.074E-4)	(0.039E-4)
SUL90P	135E-4	117E-4	191E-4	89.8E-4	231E-4	128E-4	219E-4	−156E-4	95.3E-4
	(45.9E-4)[c]	(54E-4)[b]	(87E-4)[b]	(53E-4)	(87.8E-4)[c]	(90E-4)	(195E-4)	(217E-4)	(65.7E-4)
SUL90P2	−5.54E-4	−4.81E-4	−7.91E-4	−3.67E-4	−9.36E-4	−5.46E-4	−11.6E-4	5.76E-4	−3.38E-4
	(1.72E-4)[c]	(2.0E-4)[b]	(3.32E-4)[b]	(1.97-4)	(3.24E-4)[c]	(3.3E-4)	(7.5E-4)	(9.9E-4)	(2.2E-4)
AGE	−0.0185	−0.0165	−0.0252	−0.022	−0.010	d	e	−0.0207	−0.0164
	(0.0034)[c]	(0.0042)[c]	(0.0057)[c]	(0.0037)[c]	(0.0071)			(0.0047)[c]	(0.0048)[c]
AGE2	7.53E-4	5.14E-4	14.9E-4	8.99E-4	3.32E-4	d	e	8.71E-4	6.4E-4
	(2.44E-4)[c]	(3.0E-4)	(4.2E-4)[c]	(2.66E-4)[c]	(5.27E-4)			(3.47E-4)[c]	(3.46E-4)
CHESTINF	0.0475	0.0495	0.044			0.071	0.103	0.046	0.048
	(0.010)[c]	(0.012)[c]	(0.018)[c]			(0.019)[c]	(0.042)[b]	(0.014)[c]	(0.015)[c]
CHRON	0.044	0.0390	0.052			0.036	0.003	0.036	0.052
	(0.0059)[c]	(0.0071)[c]	(0.011)[c]			(0.011)[c]	(0.024)	(0.0083)[c]	(0.0084)[c]
CROWD	0.0184	0.0163	0.018	0.019	0.011	0.037	0.029	0.022	0.0156
	(0.0073)[b]	(0.0090)	(0.013)	(0.0083)[b]	(0.014)	(0.017)[b]	(0.715)	(0.010)[b]	(0.010)
EDU	−0.0012	−0.0045	0.0056	−0.0016	−0.0060	−0.0029	−0.0031	−0.00028	−0.0024
	(0.0021)	(0.0028)	(0.0039)	(0.0025)	(0.0095)	(0.0046)	(−0.315)	(0.0031)	(0.0032)
EPIDEM	0.072	0.089	0.036	0.055	0.099	0.064	0.059	−0.23	0.098
	(0.011)[c]	(0.013)[c]	(0.019)	(0.012)[c]	(0.021)[c]	(0.022)[c]	(1.31)	(0.085)[c]	(0.017)[c]
SEX1F	0.0076	0.0083	0.006	0.0066	0.011	−0.0080	0.0027	0.0021	0.012
	(0.0052)	(0.0063)	(0.0092)	(0.0058)	(0.010)	(0.011)	(0.023)	(0.0072)	(0.0071)
SMKPPD	−0.0013		0.0019	−0.0027	0.0021	−0.0050	−0.0058	0.00126	−0.0037
	(0.0020)		(0.0049)	(0.0023)	(0.0036)	(0.0040)	(0.0086)	(0.0028)	(0.0028)
GAS	−0.020	−0.044	0.012	0.0044	−0.061	−0.057	−0.070	−0.0012	−0.036
	(0.012)	(0.015)[c]	(0.019)	(0.014)	(0.021)[c]	(0.026)[b]	(0.061)	(0.016)	(0.016)[b]
RAIN	−0.0056	−0.0068	−0.0027	−0.0050	−0.0060	−0.0054	0.0019	−0.019	−0.0027
	(0.0016)[c]	(0.0019)[c]	(0.0027)	(0.0017)[c]	(0.0031)	(0.0032)	(0.0064)	(0.0053)[c]	(0.0020)
TEMP	0.0022	0.0021	0.0025	0.0020	0.0026	0.0029	0.0015	0.0018	0.0026
	(0.00041)[c]	(0.00048)[c]	(0.00072)[c]	(0.00045)[c]	(0.00081)[c]	(0.00082)[c]	(0.0018)	(0.00086)[b]	(0.00066)[c]
RACE1W	0.056	0.039	0.073	0.051	0.060	0.059	0.087	0.034	0.075
	(0.0090)[c]	(0.012)[c]	(0.14)[c]	(0.0095)[c]	(0.019)[c]	(0.017)[c]	(0.039)[b]	(0.013)[c]	(0.013)[c]
N	16474	11497	4977	11557	5246	5108	1387	8176	8298
F	25.5	19.7	8.83	15.29	8.33	5.68	1.84	8.37	22.5
R^2	0.0286	0.030	0.033	0.022	0.026	0.027	0.025	0.019	0.049

[a] Standard errors in parentheses.
[b] Significant at the 5% level.
[c] Significant at the 1% level.
[d] AGE and AGE2 were replaced by dummy variables AGEONE (= 1 if AGE = 1, 0 otherwise) and AGETWO.
[e] AGE and AGE2 were replaced by dummy variables AGEONE through AGESIX.

whether the child is reported ill during the two-week period in question, and duration of restricted activity (RADS), which takes an integer value between 0 and 14.

Tables I and II provide descriptive statistics on these variables. Note the low number for mean RADS, indicating the large percentage of observations with a zero value for this variable. Correlation coefficients between each of the pollutants and temperature are also provided. Note that correlations between pollutants are all quite low. We searched for more complicated patterns of collinearity by using the diagnostic tests[18] provided with the SAS regression package. These tests failed to reveal any serious collinearity problems involving any of the independent variables.

We relied primarily on a linear probability model for our analysis, using ordinary least squares (OLS) as the estimation procedure, the results of which are presented below. However, the OLS model requires a number of assumptions of questionable validity for the current problem. We discuss later the effects of these assumptions on the outcomes.

Results

Table III shows the results of the regressions predicting illness incidence. Column A gives the results for the entire sample of children aged 12 and under. The remaining columns show results for a number of subpopulations; we used these results to examine the stability of the coefficients and to identify populations especially sensitive to the pollution variables. Thus, Columns B and C give results for children of mothers who do and do not smoke, and Columns D and E give results for children with and without chronic respiratory disease or a history of respiratory ailments. In Columns F and G we examine the illness incidence in younger children. Finally, in Columns H and I we divide the sample into fall (October–December 1972) and spring (January–April 1973) time periods.

The specification of the NO_2 variable was piecewise linear, with a break at 100 $\mu g/m^3$. This specification was the best of all those examined. In Table III NO2MAXL and NO-

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2MAXH refer, respectively, to average daily maximum concentrations below and above 100 $\mu g/m^3$. As shown, for NO2MAX concentrations below 100 $\mu g/m^3$, illness probability falls relatively sharply as NO2MAX increases. Above 100 $\mu g/m^3$, illness probability gradually increases with NO2MAX, but the illness rate at the highest observed two-week maximum concentration (384 $\mu g/m^3$) is less than that at the lowest observed concentration (27 $\mu g/m^3$). The clinical literature gives no reason why a dose-response function should have these characteristics.

In the subsamples (Columns B to I), the above relationship between NO2MAX and illness is replicated for children with nonsmoking mothers and children without a history of respiratory disease. For both preschool age children and infants, the coefficients were similar although not always significant. However, for children whose mothers smoke or have a chronic respiratory condition, NO2MAXH has virtually no effect. Finally, when only fall periods are examined, NO2MAX is not related to illness at all.

These exceptions did not increase our confidence in the results. The first two exceptions suggest that a "sensitive population" for NO_2 is healthy older children of nonsmoking mothers. If so, perhaps the presence of a chronic condition swamps the small NO_2 effect. Likewise, perhaps for children exposed to parental smoking an additional NO_2 effect cannot be detected. One problem with this explanation is that we found no adverse effect of mother's smoking on children's health. As for the absence of an NO_2 effect in the fall, we note that the prevalence of illness in that season was relatively low in any event. If the effect of NO_2 is to reduce resistance to disease, we might expect to find no NO_2 effect when little disease is present in the community. Such speculation notwithstanding, we have not found an effect from NO_2 that is supported by clinical evidence or that is present in all population subgroups.

For sulfates and particulates the best fits were obtained for the 90th percentile of two-week concentration and a quadratic specification, with a positive linear and negative square term. The particulate results were reasonably consistent across subpopulations, but rarely significant at the 5% level. Moreover, the various functions were such that the effects of particulates on illness were negative at concentrations below 80–100 $\mu g/m^3$, which is near the average 90th percentile concentration. That is, over much of the relevant range the particulate variable is inversely related to illness.

The coefficients for sulfates are significant for the entire sample, but not for the fall and spring semesters separately. For fall, the coefficients enter with signs and reverse of all other subsamples, but the t-values are very small. For spring, the coefficients are similar to coefficients in other equations but the t-values still are not significant. For the population subsamples the sulfate coefficients are stable and significant except for infants, where, as we have noted, sample sizes are much smaller and an effect of population on health would be correspondingly more difficult to identify. The inconsistent seasonal results may be related to using weighted averages of monthly summaries of daily readings instead of two-week averages, which were unavailable for particulates and sulfates.

Turning briefly to the other explanatory variables, the most statistically significant and robust results were for variables that one would expect to be associated with respiratory disease: age, a history of chest infection, presence of a chronic condition, the extent of crowding in the home, and outside temperature. Not only were these variables almost always significant, but the coefficients were stable across subpopulations. The coefficients for CHRON (presence of chronic disease), for example, varied between 0.036 and 0.052, except in the equation for infants, and indeed very few infants in the sample were diagnosed for a chronic disease. The EPIDEM variable was also generally significant but in the fall the sign was negative, a result we believe to be fortuitous, inasmuch as the variable was very small in absolute value during that season.

For two other variables, RACE1W and RAIN, the results were stable and significant. White children consistently reported more new illness than nonwhites. We also found a consistent inverse relationship between the amount of rain-

Table IV. Comparison of specifications of NO_2 variables in equations predicting illness incidence[a]

	A	B	C	D	E	F	G
NO2MAX	1.4E-6 (56.8E-6)	−3.5E-4 (1.82E-4)[b]	−21.7E-4 (4.37E-4)[c]				
NO2MAX2		0.009E-4 (0.0045E-4)	11.3E-6 (2.31E-6)[c]				
NO2MAX3			−1.59E-8 (0.35E-8)[c]				
NO2MAX(0–75)				−19E-4 (4.63E-4)[c]	−21E-4 (4.88E-4)[c]		
NO2MAX(75–150)				−0.16E-4 (0.94E-4)			
NO2MAX(75–100)					−0.28E-4 (0.80E-4)		
NO2MAX(100–150)					1.2E-4 (1.1E-4)		
NO2MAX(>150)				1.7E-4 (1.15E-4)	0.56E-4 (1.19E-4)		
NO2MAX(0–100)						−8.9E-4 (2.05E-4)[c]	
NO2MAX(>100)						1.7E-4 (0.69E-4)[b]	
NO2AVG(0–50)							−9.4E-4 (4.1E-4)[b]
NO2AVG(>50)							1.96E-4 (3.8E-4)
F	25.8	24.7	24.5	24.0	23.2	25.5	24.7
R^2	0.0274	0.0277	0.0289	0.0281	0.0288	0.0286	0.0278
N	16474	16474	16474	16474	16474	16474	16474

[a] Standard errors in parentheses.
[b] Significant at the 5% level.
[c] Significant at the 1% level.

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Table V. Cross section time series regression coefficients: illness incidence and restricted activity days, full sample vs. sample consisting of one child per family.[a]

Variable	Illness incidence		Restricted activity days	
	Full sample	Sub-sample	Full sample	Sub-sample
Intercept	0.0512	−0.0016	0.348	0.275
	(0.06)	(0.73)	(0.135)[b]	(0.20)
NO2MAXL	−8.87E-4	−7.98E-4	−25.0E-4	−28.7E-4
	(2.05E-4)[c]	(2.9E-4)[c]	(5.6E-4)[c]	(8.3E-4)[c]
NO2MAXH	1.71E-4	1.39E-4	4.4E-4	6.22E-4
	(0.68E-4)[b]	(0.99E-4)	(1.86E-4)[b]	(2.8E-4)[b]
PAR90P	8.88E-4	17.8E-4	−18.3E-4	9.80E-4
	(6.08E-4)	(8.8E-4)[b]	(16.6E-4)	(24.5E-4)
PAR90P2	−0.053E-4	−0.068E-4	0.064E-4	−0.04E-4
	(0.028E-4)	(0.040E-4)[b]	(0.075E-4)	(0.099)
SUL90P	135E-4	107E-4	212E-4	73.3E-4
	(45.9E-4)[c]	(66E-4)	(126E-4)	(185E-4)
SUL90P2	−5.54E-4	−4.73E-4	−9.3E-4	−4.26E-4
	(1.72E-4)[c]	(2.5E-4)	(4.7E-4)[b]	(6.9E-4)
AGE	−0.0185	−0.020	−0.045	−0.044
	(0.0034)[c]	(0.0049)[c]	(0.0092)[c]	(0.014)[c]
AGE^2	7.53E-4	8.54E-4	20.9E-4	19.1E-4
	(2.44E-4)[c]	(3.6E-4)[b]	(6.7E-4)[c]	(10.0E-4)
CHESTINF	0.0475	0.053	0.178	0.196
	(0.010)[c]	(0.015)[c]	(0.028)[c]	(0.042)[c]
CHRON	0.044	0.047	0.099	0.110
	(0.0059)[c]	(0.0084)[c]	(0.016)[c]	(0.023)[c]
CROWD	0.0184	0.018	0.019	0.0080
	(0.0073)[b]	(0.010)	(0.020)	(0.026)
EDU	−0.0012	0.0028	−0.0087	−0.0023
	(0.0021)	(0.0032)	(0.0061)	(0.0088)
EPIDEM	0.072	0.080	0.228	0.221
	(0.011)[c]	(0.015)[c]	(0.029)[c]	(0.042)[c]
SMKPPD	−0.0013	−0.0039	0.0004	0.0086
	(0.0020)	(0.0028)	(0.0050)	(0.0078)
GAS	−0.020	−0.0021	−0.037	0.0021
	(0.012)	(0.0016)	(0.032)	(0.045)
RAIN	−0.0056	−0.0049	−0.0037	0.0025
	(0.0016)[c]	(0.0022)[b]	(0.0043)	(0.0061)
TEMP	0.0022	0.0020	0.0029	0.0030
	(0.0004)[c]	(0.00057)[c]	(0.0011)[c]	(0.0016)
RACE1W	0.056	0.065	0.127	0.136
	(0.0090)[c]	(0.014)[c]	(0.025)[c]	(0.038)[c]
SEX1F	0.0076	0.0094	0.023	−0.0039
	(0.0052)	(0.0074)	(0.014)	(0.021)
N	16474	8158	16474	8158
F	25.5	13.6	20.5	10.6
R^2	0.0286	0.031	0.023	0.024

[a] Standard errors in parentheses.
[b] Significant at the 5% level.
[c] Significant at the 1% level.

fall and the incidence of illness in a two-week period, although the magnitude varied by a factor of six between fall and spring. Again, we have no explanation for this result. In addition, the presence of a gas stove in the house appeared to be unrelated to disease incidence. Few significant results were obtained, and for those that were significant the sign was contrary to expectation. As only 5% of the households cooked with gas, these generally inconsistent results are not particularly surprising.

Other covariates had virtually no explanatory power, and were rather unstable across subpopulations: educational level of head of household, sex, and mother's smoking status. In particular, we found that a mother's smoking in the home was unrelated to acute respiratory disease incidence of her children. However, this should not be too surprising in view of the contradictory findings on the health effects of passive smoking.[19]

An analysis was also carried out for illness duration as the dependent variable. In this case the dependent variable S_{ijt} took an integer value between 0 and 14. OLS estimates predicting illness duration were very similar to the results presented above; that is, independent variables that were also significant and [illegible]. However, OLS estimates of truncated variables are inconsistent as well as inefficient,[20] so it is especially important to compare the results to those of a more suitable estimation procedure. Thus, illness duration was also investigated using Poisson regression, and the comparison between Poisson and OLS is discussed below.

Some Problems of Estimation

A pervasive problem in the estimation of the effects of air pollution on illness is that information on personal exposure to pollutants is rarely available. Researchers have been obliged to use ambient monitoring data as a proxy for personal exposure, and our study is no exception to this rule. Nonetheless, every child in our sample lived and attended school within a mile of a monitoring site, a relatively tight radius compared to most similar studies.

Besides this measurement difficulty, there were several major econometric problems. These problems arose primarily from our desire to use a linear probability model and OLS as the principal estimation procedure. Convenient though it may be, the OLS model requires a number of assumptions of questionable validity for the current problem. The question we now examine is whether these refinements make much difference to outcomes.

The first problem is that the dependent variable S_{ijt} is limited to the values 0 or 1 (for illness incidence) or to the small positive integers (for illness duration). Thus, the OLS estimators are not efficient, and the linear probability model may not be appropriate in any event.

A second problem is concerned with the functional form of the relationship between illness and air pollution (indeed, between illness and any explanatory variable). As there is no theory to guide the selection of functional form, we chose a functional form on the basis of an information criterion proposed by Sawa.[21]

The third problem involves the structure of the disturbance term ϵ_{ijt}. We examined two alternatives to the OLS assumption of uncorrelated disturbances:

Autoregression: an individual's health status in one period may affect his or her health status in subsequent periods, in which case $E(\epsilon_{ijt}\epsilon_{ijt'}) \neq 0$ for $t \neq t'$.

Contagion: one's health may be affected by the health of others, especially family members and classmates, in which case $E(\epsilon_{ijt}\epsilon_{i'j't}) \neq 0$ for $i \neq i'$ or $j \neq j'$.

These problems were examined sequentially. First, several alternative functional forms were examined. Having selected a functional form, we then examined the error structure. Finally, alternative estimation procedures more suited to limited dependent variables were investigated.

Functional Form

Table IV shows the relationship between illness incidence and NO_2 for several different specifications of the pollution variable. The basic variable was NO2MAX, the daily maximum NO_2 reading, averaged over the two-week period. (Not shown are specifications using average pollution variables, which give results inferior to the ones for NO2MAX.)

The specifications examined include the following:

- linear specification
- quadratic
- cubic
- piecewise linear functions with one break point at 100 $\mu g/m^3$, two break points at 75 and 150 $\mu g/m^3$, and three break points at 75, 100 and 150 $\mu g/m^3$.

In all specifications, except the linear, the relationship between NO_2 and illness incidence is U-shaped. Based on

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Table VI. Comparison of logit and OLS models predicting illness incidence.

	OLS		Logit		
	Coefficient	Std. error	Coefficient	Std. error	$\frac{\partial S}{\partial x}$
Intercept	0.176	0.029[b]	−1.80	0.256	
NO2MAXL	−8.98E-4	2.04E-4[b]	−0.0067	0.00174[b]	−7.63E-4[b]
NO2MAXH	1.61E-4	0.68E-4[a]	0.00135	0.000595[a]	1.53E-4[a]
PAR90P	−2.63E-4	0.923E-4[a]	−0.00187	0.00082[b]	−2.13E-4[a]
SUL90P	2.55E-4	12.1E-4	−0.00138	0.0108	−1.57E-4
AGE	−0.0082	0.00085[b]	−0.0715	0.0074[b]	−0.00814[b]
CROWD	0.150	0.0071[a]	0.151	0.064[a]	0.0172[a]
EPIDEM	0.074	0.010[b]	0.537	0.087[a]	0.061[b]
GAS	−0.020	0.012	−0.187	0.110	−0.020
RAIN	−0.0063	0.0016[b]	−0.051	0.015[b]	−0.0058[b]
TEMP	0.00184	0.00039[b]	0.0167	0.00356[b]	0.00190[b]
RACE1W	0.0540	0.0088[b]	0.542	0.093[b]	0.0528[b]
CHRON	0.0416	0.0059[b]	0.350	0.0450[b]	0.0423[b]
CHESTINF	0.0460	0.0102[b]	0.342	0.081[b]	0.0434[b]

[a] Significant at the 5% level.
[b] Significant at the 1% level.

the BIC criterion proposed by Sawa,[21] the best performer is the piecewise linear specification with a break point at 100 $\mu g/m^3$.

We also tested these spline specifications against the quadratic specification using one of the tests described by Davidson and MacKinnon[22] for non-nested models. The result of this test was as follows: When the quadratic specification was taken as the null hypothesis against the piecewise linear alternative, the null hypothesis was rejected. However, with the spline taken as the null hypothesis the null could not be rejected. Thus, the spline specification with a break point at 100 $\mu g/m^3$ fit the data best, and this was used in subsequent work.

Error Structure

To examine the effect of possible serial correlation we assumed a first-order autocorrelation scheme and used a two-stage procedure described by Kmenta.[23] First we estimated the autocorrelation parameter ρ using OLS, and then reestimated the model

$$(Y_t - \hat{\rho} Y_{t-1}) = (X_t - \hat{\rho} X_{t-1})\beta + (\epsilon_t - \hat{\rho}\epsilon_{t-1}).$$

Our estimate for ρ was $\hat{\rho} = 0.036$. In the second stage, we found the following results for the NO_2 variables, which, it will be noted, are essentially the same as Column H of Table III:

$$S = \underset{(2.12E-4)}{-9.35E-4\ NO2MAXL} + \underset{(0.81E-4)}{1.76E-4\ NO2MAXH} + \text{other terms}$$

with $n = 14907$ and $F = 25.1$ for the equation. This result indicated that the problem of autocorrelation could be ignored.

Contagion presented a problem that we were not able to resolve fully, due to a lack of complete information on all the physical contacts among the various members of the sample. However, we were able to examine contagion in the home, one of the most likely places where diseases may be spread.

If contagion in the home is present, the estimated effect on incidence and duration of variables common to members of a family, such as their exposure to air pollutants, will exceed the true effect. To test for this possibility we compared regression results from the full sample to the results from a subset consisting of one child chosen randomly from each family represented in the sample (Table V). Although the standard errors on the former are a bit larger, (which is what one would expect from the reduction in sample size), the coefficients are quite similar. Thus, the results are probably not much affected by spread of disease in the home.

Limited Dependent Variables

In this section we examine whether the results depend on our use of OLS rather than techniques more suited to limited dependent variables. Specifically, we tested the linear probability model against a logit model for predicting illness incidence, using the "C" test described by Davidson and MacKinnon.[22] This test showed the logit model to be superior in the following sense: When the null hypothesis H_0 is the

Table VII. Comparison of Poisson and OLS models predicting illness duration.

	OLS		Poisson		
	Coefficient	Std. error	Coefficient	Std. error	$\frac{\partial S}{\partial x}$
Intercept	0.352	0.079[b]	−1.62	0.361[b]	
NO2MAXL	−0.00249	0.00056[b]	−0.0083	0.0023[b]	−0.00173[b]
NO2MAXH	0.00043	0.000186[a]	0.00195	0.00084[a]	0.00041[a]
PAR90P	−0.0047	0.00025	−0.00101	0.00120	−0.0021
SUL90P	−0.0026	0.0033	−0.0120	0.016	−0.0025
AGE	−0.0174	0.0023[b]	−0.078	0.0102[b]	−0.0162[b]
CROWD	0.0089	0.0193	0.075	0.092	0.0156
EPIDEM	0.216	0.028[b]	0.768	0.117[b]	0.160[b]
GAS	−0.032	0.031	−0.155	0.158	−0.030
RAIN	−0.0044	0.0042	0.0022	0.0195	0.00046
TEMP	0.0030	0.0011[b]	0.0152	0.0052[b]	0.0032[b]
RACE1W	0.119	0.024[b]	0.688	0.148[b]	0.114[b]
CHRON	0.091	0.016[b]	0.399	0.069[b]	0.093[b]
CHESTINF	0.178	0.028[b]	0.571	0.097[b]	0.155[b]

[a] Significant at the 5% level.
[b] Significant at the 1% level.

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OLS model and the logit model is H_1, H_0 is rejected; however, when the roles are reversed and H_0 is the logit model, H_0 cannot be rejected.

For illness duration a similar comparison was made between OLS and Poisson regression. Again, the OLS model was found to be inferior. Nonetheless, the coefficients on the independent variables estimated using OLS were very similar to the corresponding coefficients for the logit and Poisson models. These coefficients are compared in Tables VI and VII. To facilitate comparison, the rightmost column of each table is the derivative of the dependent variable of the Poisson or logit function, evaluated at the mean of the dependent variable. (For discrete independent variables the entry is the average change in probability of illness, estimated by the weighted sum of the change in probability when the variable is added at the mean and when it is taken away.) Even though OLS appeared slightly inferior to logit and Poisson regression in predicting illness incidence and duration, the qualitative results were hardly affected.

Conclusions

A CHESS data base from Chattanooga, Tennessee was thoroughly scrutinized and found to be of high enough quality to warrant epidemiological analysis. Using this data base, the relationship between NO_2 ambient pollution levels and acute respiratory disease in children was examined. Although a statistically significant relationship was found, it was not monotonic. Indeed, over the range of pollution values experienced, more illness is associated with low pollution values than with high ones. A U-shaped relationship between illness and NO_2 concentrations was found in several subpopulations in addition to the entire data set, although for some subpopulations no relationship was found. As far as we know, there is no clinical explanation for this result. In contrast, higher ambient sulfate levels were found to have a positive effect on acute respiratory disease incidence in children over the entire period and for different subsamples, although this effect was not significant for either season analyzed separately.

The strange relationship between NO_2 concentrations and ARD in children could be attributable to three problems inherent in any epidemiological study. First, the relationship could be entirely fortuitous, although the odds against this for our study are long. Second, both illness and NO_2 could be related to some unobserved variable. However, such a variable must have strange properties, because for certain well-defined subsets, its relationship to either illness or NO_2 changes substantially. Finally, the data could still contain biases that create the observed effects.

In short, there is reason to be skeptical of a U-shaped dose-response function relating ambient NO_2 levels and acute respiratory disease. Nonetheless, we suggest that nonmonotonic dose-response functions be explicitly considered in future epidemiological or clinical research on the health effects of NO_2 and perhaps other pollutants as well.

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